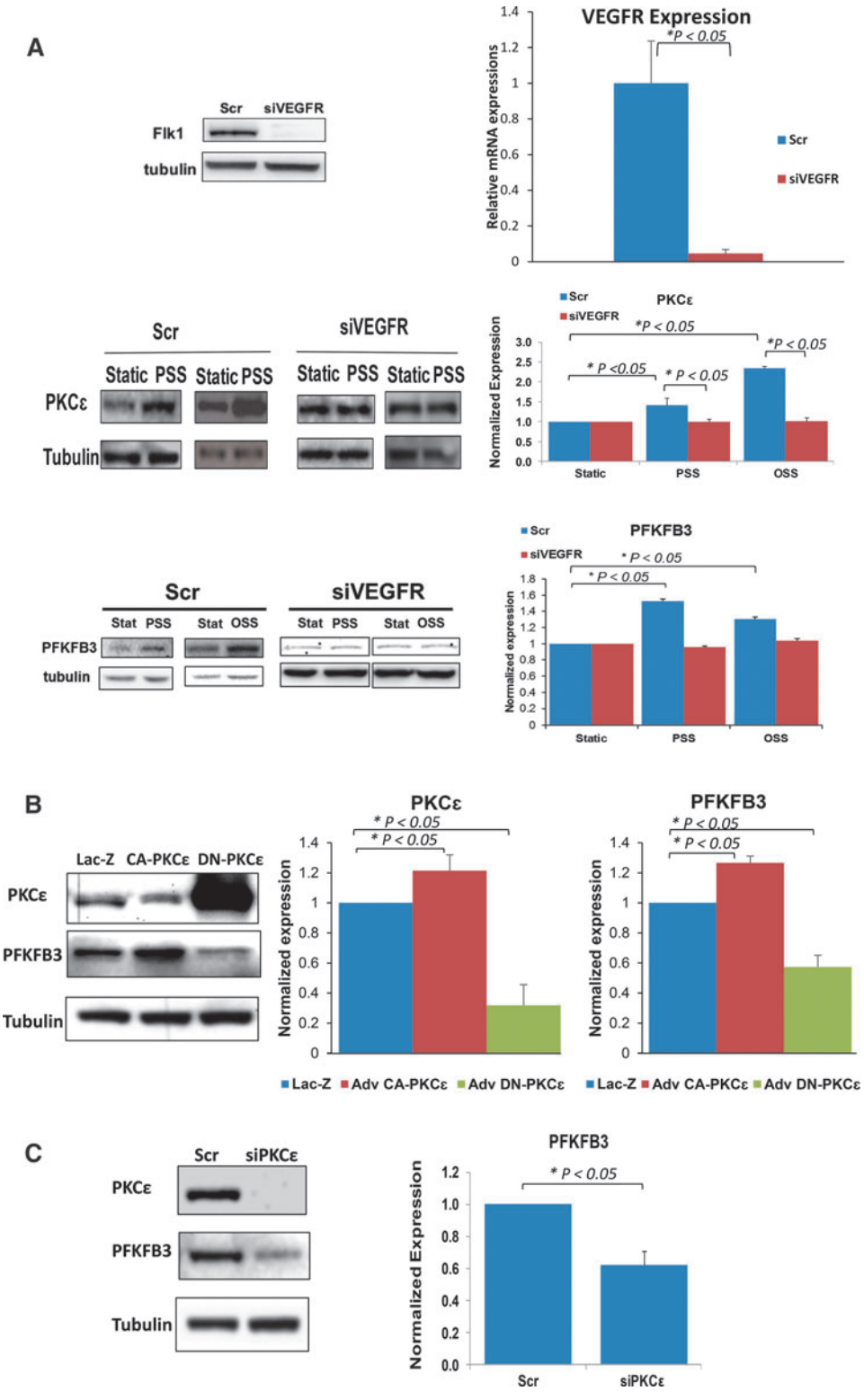
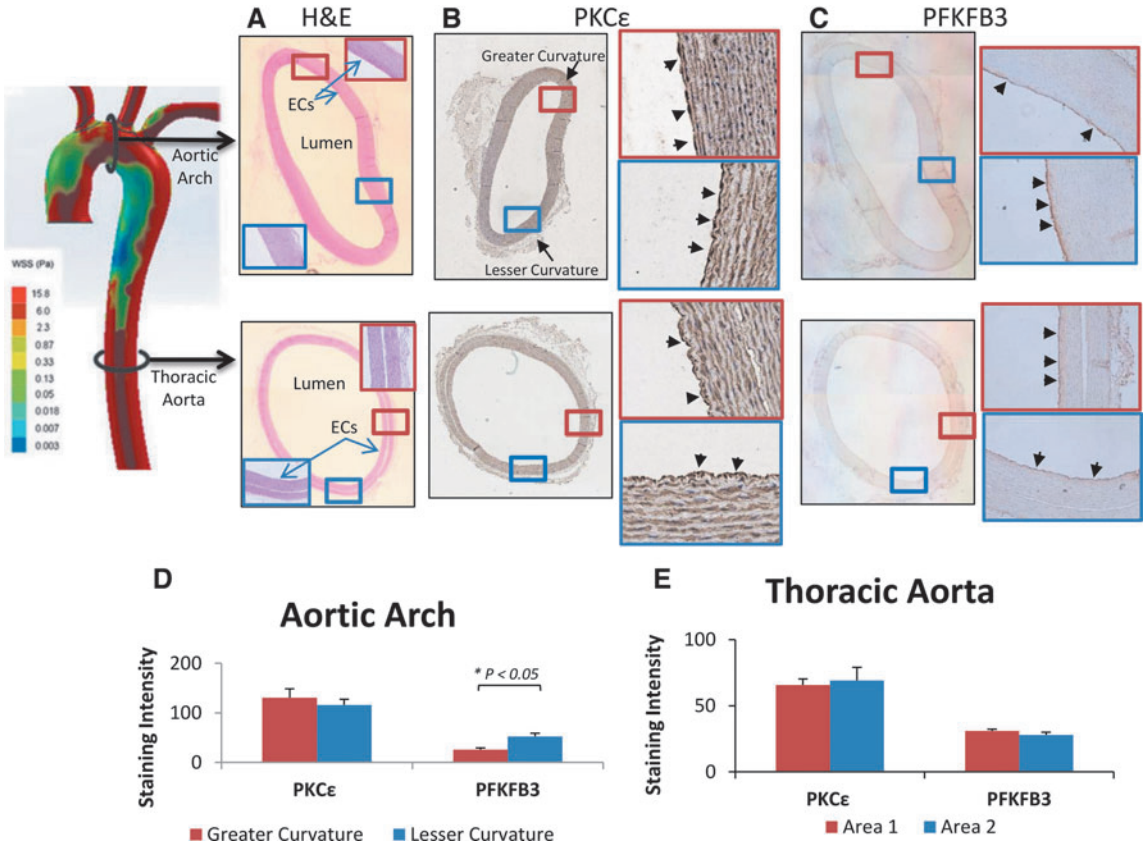
Supplementary Data





SUPPLEMENTARY FIG. S2. PKCε and PFKFB3 immunostaining. (A) ECs were stained with H&E in the cross-sections of NZW rabbit aortic arch and thoracic aorta. **(B)** PKCε and **(C)** PFKFB3 immunostaining was prominent in the endothelial lining (*black arrows*). **(D)** In the aortic arch, PFKFB3 were differentially stained at lesser and greater curvatures (*red vs. blue*) (*$p < 0.01$, $n = 3$). **(E)** In the thoracic aorta, PKCε and PFKFB3 were equally stained. ECs, endothelial cells; H&E, hematoxylin and eosin; NZW, New Zealand White.

SUPPLEMENTARY FIG. S1. Shear stress-induced VEGFR-dependent PKCε expression. (A) HAEC were transfected with scrambled (Scr) siRNA or VEGFR2 siRNA (siVEGFR) and were subjected to three conditions (i) static, (ii) PSS, and (iii) OSS. The density quantification of Western blots was normalized to β-Tubulin. PSS and OSS differentially increased VEGFR2-dependent PKCε protein expression (*$p < 0.05$, $n = 4$). **(B)** HAEC were infected with recombinant Adenoviruses LacZ, CA-PKCε, or DN-PKCε. CA-PKCε promoted, whereas DN-PKCε reduced PFKFB3 protein expression (*$p < 0.05$ *vs.* control, $n = 4$). **(C)** HAEC were transfected with scrambled (Scr) siRNA or siPKCε to assess PKCε-dependent PFKFB3 expression. siPKCε mitigated shear stress-induced PFKFB3 protein expression (*$p < 0.05$, $n = 3$). CA, constitutively active; DN, dominant negative; HAEC, human aortic endothelial cells; OSS, oscillatory shear stress; PFKFB3, 6-phosphofructo-2-kinase/fructose-2,6-biphosphatase 3; PKCε, protein kinase C isoform epsilon; PSS, pulsatile shear stress; siRNA, small interfering RNA; VEGFR, vascular endothelial growth factor receptor.